864 Electrophysiology of Atrial Arrhythmias

Tuesday, March 31, 1998, 4:00 p.m.–5:30 p.m.
Georgia World Congress Center, Room 254W

4:00

864-1 Importance of Refractoriness Heterogeneity in Electrical Remodeling by Atrial Tachycardia

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Background: Atrial tachycardia, such as occurs during atrial fibrillation (AF), is known to cause electrophysiological changes which promote the occurrence and maintenance of AF. This study investigated the role of refractoriness heterogeneity in determining the duration of induced AF (DAF) in 12 control dogs (CD) and 11 dogs paced at 400/min for 24 h (PD).

Methods: DAF was measured as the average of 10 trials/dog. Epicardial arrays containing 240 bipolar electrodes for stimulation and/or recording were attached to cover the entire surface of both atria. Atrial effective refractory period (ERP) was measured at a cycle length of 300 ms at a large number of atrial epicardial sites per dog (65 ± 5, CD, M ± SE, 68 ± 7, PD), and the coefficient of variation in ERP (CVR = SD/M · 100) was used as an index of ERP variability.

Results: PD had significantly greater DAF (153 ± 53 s) than CD (39 ± 28 s, $^{*}p < 0.05$). Pacing reduced ERP (from 120 ± 4 to 104 ± 2 ms, $^{**}p < 0.01$) and increased CVR (from 15 ± 1 to 21 ± 1%**). Increased CVR in PD was due both to regional differences in the extent of ERP remodeling (significant in right atrial appendage, free wall, and left appendage = 21 ± 17**, 20 ± 14**, 15 ± 10 ms* decreases vs CD respectively; NS in Bachmann's bundle and posterior left atrium) and to increased intersite variability within regions. When both ERP and CVR were included in a multilinear regression model for DAF, CVR was a highly significant predictor of DAF ($p < 0.0001$), but ERP had only marginal predictive value ($p = 0.04$).

Conclusions: Twenty-four hours of rapid atrial activation increases ERP variability both among and within various atrial regions. Increased ERP variability seems to be more important than the decrease in ERP for promoting the maintenance of AF in tachycardia-remodeled atria.

4:15

864-2 Recovery From Pacing-induced Heart Failure Is Associated With Persistence of Triggered Atrial Tachycardia in Spite of Normalization of Atrial Refractoriness and Ventricular Function

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We recently showed that dogs with rapid ventricular pacing-induced heart failure (CHF) have: 1) Reverse-use dependent prolongation of atrial effective refractory period (ERP); 2) Inducible atrial tachycardia (AT) which based on responses to pacing, drugs (verapamil and flunarizine), and mapping have a focal mechanism presumably related to triggered activity due to Ca^{2+} overload. To evaluate the atrial electrophysiological recovery from CHF, ventricular pacing (235 bpm) was discontinued in 6 CHF dogs paced for 3–5 weeks. Atrial ERP and AT inducibility were assessed weekly in the conscious state with 2–3 transvenous, chronic right atrial leads. During 10–35 days of recovery (mean 22 ± 11 days), CHF resolved and LVEF normalized. Sustained AT (~10 min) - which was not inducible at baseline but was easily induced during CHF- remained inducible in 6/6 dogs during the entire recovery phase. However, atrial ERP prolongation noted during CHF normalized (baseline, 120 ± 8 ms; CHF, 148 ± 20 ms; recovery, 126 ± 20 ms). Both during CHF and recovery, AT was induced with burst atrial pacing, entrainment did not occur, and AT cycle length was similar (CHF, 125 ± 6 ms; recovery, 124 ± 16 ms; p = NS). Verapamil (3 ± 1 mg) terminated AT during CHF and recovery. Ryanodine (9 ± 1 μg/Kg), a Ca^{2+} overload blocking drug which was not used during CHF, terminated AT during recovery. Acute suppression of AT reinduction was noted with both drugs during CHF and recovery.

Conclusion: CHF-induced prolongation of atrial ERP is dissociated from AT inducibility. CHF episodes may predispose the atrium to persistent triggered arrhythmias due to intracellular CA^{2+} overload.

4:30

864-3 The Crista Terminalis Is Not Normally a Barrier to Conduction – Implications for Atrial Flutter

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Background: During atrial flutter (AFl) in humans, reentrant activation usually travels either up the atrial septum and down the right atrial free wall (typical AFl) or vice versa (atypical AFl). Recent mapping of AFl in humans demonstrated a line of block along the crista terminalis (CT). This line of block was thought to be anatomic (fixed).

Methods: The canine heart is a model that is extensively used to understand electrophysiologic events in humans. To test the hypothesis that block along the CT during AFl is functional, we analyzed atrial activation patterns during 1) sinus rhythm, 2) pacing (cycle lengths of 500 ms to 150 ms) from selected right atrial (RA) and left atrial (LA) sites, 3) AFl, and 4) atrial fibrillation (AFib) in 10 dogs with sterile pericarditis and 2 normal dogs. Electrograms (396 electrodes) were simultaneously recorded from the RA, the LA, and the atrial septum.

Results: Activation across the CT occurred during atrial pacing from selected sites (posterior-inferior LA, RA appendage and RA free wall), and during AFib. During AFl, functional block was present along a line roughly parallel to but not at the CT. Activation wave fronts from the RA to the LA and vice versa traveled over several routes, including Bachmann's bundle and inferior to the inferior vena cava, as well as across the CT.

Conclusions: In this model: 1) activation occurs across the CT, and 2) during AFl, a line of functional block occurs near the CT. These data suggest that block in the area of the CT during AFl in humans is functional.

4:45

864-4 Pulse Propagation Through the Crista Terminalis in Patients With Atrial Fibrillation and Atrial Flutter

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It has been demonstrated that the crista terminalis (CT) is an anatomic barrier to transversal conduction during atrial flutter. However, it remains unclear if it is fixed or functional. In addition, the conversion of atrial flutter to atrial fibrillation seems to be determined by a decrease of the length of a functional line of block in the right atrium. We hypothesized that the CT has transversal conduction capabilities, which are different in patients with sole atrial flutter, as compared to patients with atrial fibrillation. To test this hypothesis, we investigated the pulse propagation through the CT following pacing with 6 pacing cycle lengths (700–200 ms) and one extrastimulus at 10 pacing sites along the CT in 5 patients with sole atrial flutter and 5 patients with atrial fibrillation.

Results. In all patients during pacing with long pacing cycle lengths, a transversal conduction with fragmented electrograms was present. During shorter pacing cycle lengths and a decrease in the coupling interval, an indicated conduction delay and soon a conduction block with split electrograms developed. This block was primarily found in the central part of the CT. With progressive shortening of the pacing cycle length or further decrease of the coupling interval, it extended towards both ends of the CT. In patients with atrial flutter, the length of the line of block and the refractory period of the central part of the CT were significantly longer, as compared to patients with atrial fibrillation.

Conclusion: This study showed that the CT provides limited transversal conduction capabilities. Furthermore, it has been demonstrated that the conduction block during atrial flutter is the result of a rate dependent, progressive lengthened functional block in the CT. Finally, different transversal conduction capabilities at the CT may be responsible for the development of either atrial flutter or atrial fibrillation.

5:00

864-5 Ablation of Atrial Fibrillation in a Canine Mitral Valve Regurgitation-Rapid Atrial Pacing Model

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Background: There is considerable interest in the development of a catheter based method for ablation of atrial fibrillation (AF). In this study, we attempted to ablate AF with linear lesions in a model combining mitral valve regurgitation (MR) and rapid atrial pacing.

Methods: MR resulting in a 5–10 mm increase in pulmonary artery wedge pressure was induced by avulsion of the chordae tendinae of the mitral valve.

Lesions Given	Result	N
1	NSR	2
1, 2	NSR	1
1, 2, 3, 5	NSR	1
2, 3, 4, 5	NSR	1
2, 3, 4, 5, 6	Standstill	1
1, 2, 3, 4, 5, 6	Atrial Flutter	1
1, 2, 3, 4, 5, 6	AF	3